Canad. M. A. J.
Mar. 18, 1961, Vol. 84

CASE REPORT

TRIGGER WRIST WITH INTERMITTENT CARPAL TUNNEL SYNDROME

A HITHERTO UNDESCRIBED ENTITY WITH REPORT OF A CASE*

PHILIP EIBEL, M.D., C.M., *Montreal*

SINCE 1854, when Paget[32] first described this condition, which followed a fracture of the lower end of the radius, many cases of tardy median palsy or carpal tunnel syndrome have been reported in the literature. The trophic changes occurring in only the more severe cases of pressure on the median nerve were clearly alluded to in his treatise, though without an understanding of the action of the transverse carpal ligament in the pathogenesis. This was only appreciated towards the end of the first quarter of the twentieth century.[1] The concept of non-traumatic and idiopathic carpal tunnel syndrome, as distinguished from that due to injury, was even slower in developing, the first description in the literature being by Brain *et al.* in 1947.[6] As late as 1913, cases of acroparesthesia, a term coined by Schultze,[37] were ascribed to the presence of cervical ribs, and it is amusing to pore over the pages of the transactions of the Royal Society of Medicine at the time,[48] and see how many cases of this entity, which certainly must have included some cases of what we now call carpal tunnel syndrome, were so satisfactorily treated by section of cervical ribs. Thus Wormser,[50] writing in 1950, believes that many of the cases formerly ascribed to cervical ribs were in reality median nerve palsies. Beyers and Keet,[5] writing on the subject in 1956, state with some obvious amusement that cases of acroparesthesia appear to be diagnosed according to the fashion of the time. Thus the identical symptom-complex has been etiologically related to cervical ribs, the scalenus anticus syndrome, the thoracic inlet and costoclavicular syndromes. Acroparesthesia has also been explained purely as a hazard inherent in certain occupations, such as those pursued by tailors, cigar-workers and washerwomen, with the strain falling on the phylogenetically younger thenar muscles.[8] As late as 1939, this opinion was held by Wartenberg.[45] At the present time, however, the explanation is either cervical disc disease or carpal tunnel syndrome.

Hunt[16] in 1909 ascribed the median nerve disturbance to direct occupational traumata on the thenar branch of the median nerve distal to the ligament, but later,[17] probably owing to the influence of Marie and Foix, attributed it to direct pressure on the median nerve deep to the transverse carpal ligament. The latter investigators, in 1913,[27] clearly described the pathology of the condition, showing how the median nerve was strangled in its course within the carpal canal, and for the first time, suggested cutting it in an effort to preclude the development of the median nerve affection. Moersch[29] in 1938 suggested the same procedure as a treatment for median thenar neuritis, and Woltman[49] in 1941, and subsequently Zachary[51] in 1945, actually transected the ligament with complete relief of symptoms and objective signs. Since this time, the operation has become a regular part of the orthopedic surgeon's armamentarium, so that Phalen[33] in 1957 was able to report 71 cases of the idiopathic or non-traumatic type alone.

Actually, as is well known, any factor which reduces the size of the carpal canal as it conveys ten structures from the forearm to the palm (the flexor sublimis and profundus tendons, the flexor pollicis longus tendon, and median nerve), and/or increases the size of its contents, may give rise to the syndrome. This canal is so tight that the median nerve is normally flattened here, and mere flattening of the nerve cannot be presumed to afford proof of the syndrome.[21, 33] Diminution of the space within the canal can be caused by an enlargement of the bone and soft tissue structures forming the boundaries of the canal and/or some factor increasing its contents. Thus, it may be caused by a fracture of the lower end of the radius,[32] a dislocation of the lunate,[30] treatment of Colles' fracture in acute flexion,[28] a fracture of the os magnum,[3] acromegaly,[20, 25, 36, 49] gout,[44] occupational microtraumata,[8] osteoarthritis,[34] non-specific thickening of the flexor tendon sheaths,[31] rheumatoid arthritis,[42] amyloid disease,[13] neuroma,[10] pregnancy,[11, 43] Leri's pleonosteosis,[46] an anomalous artery,[9] thrombosis of a persisting median artery,[2] impingement of a slip of sublimis tendon,[9] carpal ganglion,[7] idiopathic nocturnal swelling,[22] non-specific tenosynovitis,[24] tuberculous tenosynovitis,[24] the shoulder-hand syndrome,[24] a plexus of varicose veins,[41] calcific tendinitis,[38] hematoma in the carpal canal,[47] sclerodactyly[19] and high frequency electric current.[18] The causes, as can readily be seen, are legion. Even the honeymoon has been incriminated.[4] In brief, any factor resulting in a disproportion between the carpal canal and its contents, owing to a decrease in the available space in the former or an increase in the volume of the latter, or both, is likely to produce the symptoms and signs of the carpal tunnel syndrome, namely, pain or tingling in the median nerve distribution of the hand and fingers, with or without paralysis of the intrinsic hand musculature innervated by the median nerve, generally the muscles of the thenar eminence. In extreme cases, trophic changes, such

*From the Section of Orthopedic Surgery, Surgical Service, Jewish General Hospital, Montreal.

as first described by Paget, will be seen. One reason for the so frequent sparing of the thenar eminence in obvious cases of median nerve disturbance is an anomalous innervation of the thenar eminence by the ulnar nerve.[15, 35] Another is the fact that motor fibres are more resistant than sensory.[26]

The author wishes to report a case which he believed erroneously to belong under the general rubric of the carpal tunnel syndrome, but which eventuated, to the best of the author's knowledge, in a hitherto undescribed though related entity.

Mrs. I.G., a white female, aged 31, was admitted to the Jewish General Hospital on July 2, 1957. Her illness began approximately four months before this date. Her complaints consisted of inability to flex completely the fingers of the right hand, weakness of the hand grip precluding pursuit of her ordinary household duties, and a tingling sensation in the thumb, index and middle fingers of the right hand, but only on complete flexion of her fingers. There was no history of a single major traumatic experience, but repeated microtraumata were accepted as an etiological factor, since, in addition to her housework, she worked as a gardener, which required the holding of heavy jars with her right hand for the purpose of replanting. It was believed at first that the affection was rheumatic in nature, though why it should involve the right side only in a generalized disease was somewhat difficult to explain. Sedimentation tests also, repeated twice, afforded normal readings.

Accordingly, the patient was treated for traumatic tenosynovitis by heat, massage, sedation and eventually immobilization in a cast for several weeks, all without a modicum of success.

Upon removal of the cast shortly before admission to the hospital, a tiny swelling was first noted which appeared just proximal to the distal volar crease of the wrist. This swelling was definable only with difficulty, and its appearance only on extreme flexion of the fingers was accompanied by a palpable click which the patient claimed was exquisitely painful. This was accompanied by tingling in the ring and index fingers, and, to a lesser extent, in the tip of the thumb. Once the swelling appeared, there was no longer any pain and tingling. When attempts were made to straighten the fingers before complete extension, the swelling above the wrist was no longer palpable, and before complete extension also there was another click known to the patient but impalpable to the examining hand and reported as not nearly so painful as the click on maximum flexion of the fingers. The patient found from experience that the only comfortable position for the fingers was one of complete extension and was loath to bend her fingers at any time. The tourniquet[12] and the Tinel[40] fourmillement tests were both negative. Full dorsiflexion of the wrist[6] and acute flexion[28] produced no change in symptoms and signs. There was very little if any sensory deficit involving the median fingers. There was no atrophy of the thenar muscles, but subjectively there was impairment of the strength of the right hand grip.

Although the nature of the swelling could not be ascertained at the time, it was considered that the patient's symptoms were severe enough to warrant exploration. This was carried out on July 4, 1957.

Owing to the fact that the swelling in the distal volar carpal crease appeared only on forced flexion of the fingers, it was thought advisable to perform the operation under brachial block anesthesia. With a 1% procaine block there is generally a loss of sensation but not of motor power, since the motor fibres are more resistant than the sensory.[26] However, although this was attempted, it failed, with even a slight pneumothorax resulting. Accordingly, it was decided to perform the operation under light general anesthesia, and when the stage of exposure of the flexor tendons was reached, to allow the patient to come out of the anesthetic state, and then to have her flex her fingers; but, as will be seen, this did not occur.

A Z-shaped incision centring over the palmaris longus was made over the distal volar crease, the two vertical limbs being about one inch and one and a half inches from the radial and ulnar extremities of the one-inch transverse component. The tissues were then deepened and divaricated, the superficial volar ligament incised, the palmaris longus tendon isolated, and the deep transverse ligament incised over a grooved director on the ulnar side of the median nerve. The nerve appeared to be flattened in its course beneath the ligament, but as noted above, this is a normal finding. Attempts were then made to have the patient flex her fingers as had been planned after the failure of the brachial block, but these were unsuccessful because the patient could not be roused from her state of anesthesia. The swelling that had been present before operation could not be discovered. The wound was then closed in layers except for the transverse ligament which was not sutured. A specimen of synovium was sent for pathological examination and was reported to consist of normal tissue.

Except for the minimal pneumothorax, the patient made an uneventful recovery, and for several weeks postoperatively there were no complaints. The patient, though encouraged to move her fingers, did not do so to any great extent owing to the usual postoperative pain, but she had none of her preoperative complaints.

About 20 days after the operation, by which time the patient had completely recovered, when she was asked to flex her fingers to the utmost, surprisingly the swelling, present prior to operation, had not only "returned" but if anything, appeared to be larger. Certainly it was now situated more distal to the original site of appearance, and it definitely was more easily definable. The swelling was tender, about one-quarter of an inch in circumference, and movable only when the fingers moved. However, owing to the fact that nothing of a pathological nature had been discovered at the time of operation, it was decided that the swelling was probably rheumatic in nature, and despite negative sedimentation tests, the patient was given injections at weekly intervals for four weeks of 1 c.c. of hydrocortisone. But she constantly, persistently and unremittingly complained of inability to flex her fingers completely, and, when she finally did succeed in approximating her fingers with her palm, there appeared the painful tumour above the wrist. Its appearance from the depths of the palm was always heralded by a painful click, easily palpable to the examining hand and audible to the patient. At the same time, there was tingling in the median distribution of the fingers. The tumour always disappeared on extension and before it disappeared was always preceded by pain,

Canad. M. A. J.
Mar. 18, 1961, Vol. 84

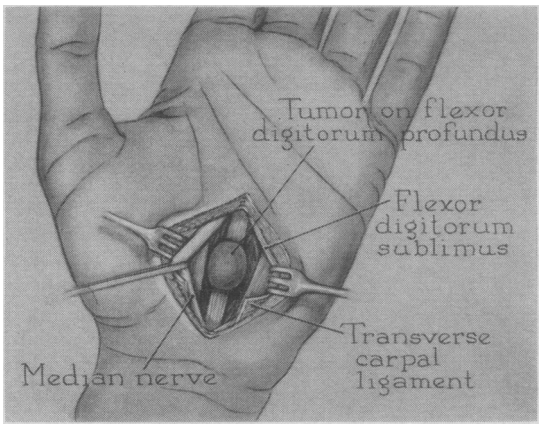

Fig. 1

though not as much as on flexion. There was now slight hypoesthesia of the middle index finger and thumb; the grip was certainly weaker than that of the opposite side; however, no palsies could be made out. Electromyographic studies were not performed.

The second operation was carried out on October 17, 1957, under 2% local infiltration of the median and ulnar nerves at the wrist. A tourniquet was then applied about the upper arm. This had not been used in the first operation since it made the procedure somewhat difficult. The median nerve was found and isolated. It appeared to be normal grossly except for the flattening mentioned in the first operation. The patient was then asked to flex her fingers, and it was realized that some of the distal fibres of the transverse carpal ligament had not been cut and that it was at this point that there appeared to be an impediment to free flexion. On extreme flexion the patient stated that "something" appeared to pass upwards as it had prior to operation, and she again had the sensation of a "click". Accordingly, with the fingers held in a position of extreme flexion, the various accessible tendons were carefully examined, and a solid oval tumour about half an inch in its greater diameter was found attached to the sheath of the flexor digitorum profundus tendon of the middle finger (see Fig. 1: artist's representation of the operative findings). The tumour was easily shelled out, and following this the patient stated that she could no longer feel the "click". The overlooked fibres of the transverse carpal ligament were then transected and the wound was closed in layers. A pressure dressing was then applied.

The wound healed by primary intention, and the "trigger" action at the wrist and the mild and intermittent signs of median neuritis have not recurred to the time of writing (two years postoperatively). The pathological report was as follows: "Specimen consists of one piece of whitish shiny tissue which is hard on palpation and roughly ovoid in shape. This measures 1.5 cm. in its greatest diameter and is covered by a thin transparent capsule. At one edge of this nodule there are several delicate fibrous tags. Upon sectioning of the nodule, the cut surface is whitish grey in colour with several yellowish areas. It is firm in consistency but shows no evidence of calcification. The microscopic section revealed a sharply circumscribed and encapsulated oval-shaped tumour composed of a small number of fibroblasts with a moderate amount of intracellular substance. The fibroblasts are quite uniform throughout and within the fibrous supporting tissue a few thin-walled blood vessels are encountered. Attached to one aspect of this tumour nodule is an irregularly shaped space lined by flattened cells similar to those making up the wall of this space. Diagnosis: Fibroma of tendon sheath."

DISCUSSION

The modus operandi of this tumour is of interest. The author found in the anatomy laboratory that the average vertical extent of the flexor retinaculum is 1" (or 2.5 cm.). A fixed point was taken in full extension of the fingers just distal to the ligament in a flexor digitorum profundus tendon and it was found that this point moved 1⅜ in. (3.34 cm.) in a proximal direction when the fingers were flexed to their fullest extent (see Fig. 2). Accordingly, a tumour attached to a profundus tendon just distal to the retinaculum would appear just proximal to it when the fingers were completely flexed and this would explain the trigger action at the wrist and pain and tingling in the median fingers due to median nerve irritation in a confined space just before the tumour appeared proximal to the retinaculum.

In full extension of the fingers, there was no pressure on the median nerve and therefore no tingling. On flexion, two phenomena occurred: first, the tumour entered the carpal canal, where it caused compression of the median nerve with tingling in the median fingers; then, after the utmost efforts at flexion, the tumour would rise proximally above the upper border of the transverse carpal ligament. At this time, the tumour would suddenly become palpable with an audible "plop" because of being freed from a tightly confined area. The tingling in the median distribution would then disappear, only to reappear temporarily during the beginning of extension of the fingers, which could only be carried out with effort needed to force the tumour through the carpal canal. Upon leaving the carpal canal the tumour no longer caused any pain or tingling.

Accordingly, there are two related entities in this case: (1) trigger or snapping wrist; and (2) directly owing to this condition, a form of median neuritis, intermittent in nature because of the intermittency of the constricting action of the tumour in passing through the carpal canal, and mild in degree because of indisposition of the patient to flex her fingers because of the resultant pain, and, in consequence, relative sparing of the median nerve.

Several other observations may be made at this juncture. The reason why the triggering and resultant intermittency of the carpal tunnel syndrome did not disappear with the first operation is simply that all the fibres of the flexor retinaculum were not cut. This is a common error in the operative procedure and many allusions are found to this effect in the literature.[14, 23, 39]

One might speculate, too, why the tumour became so much more prominent in the interval be-

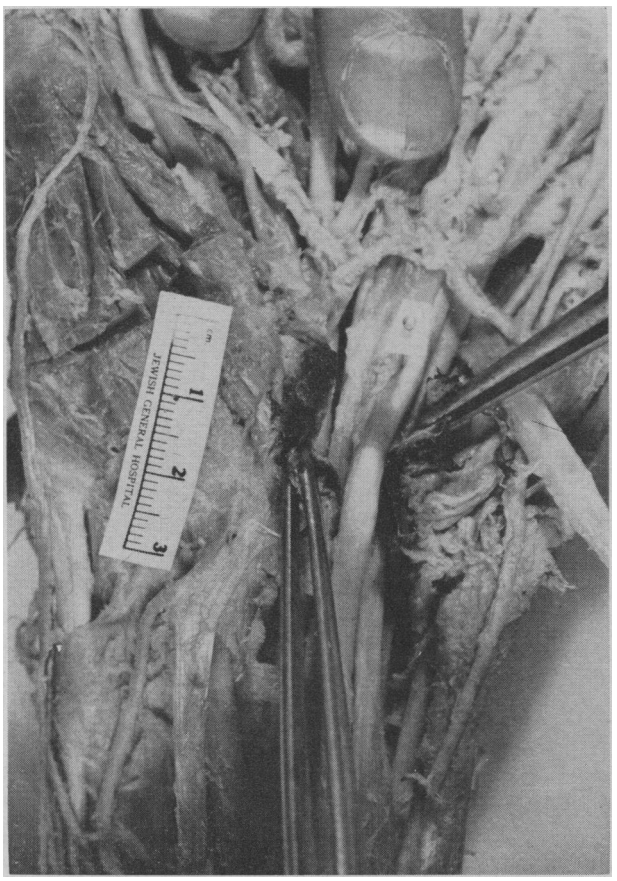

Fig. 2a (taken with fingers extended). Showing a fixed point (marked by a pin) in the flexor digitorum profundus, distal to the transverse carpal ligament (held by forceps).

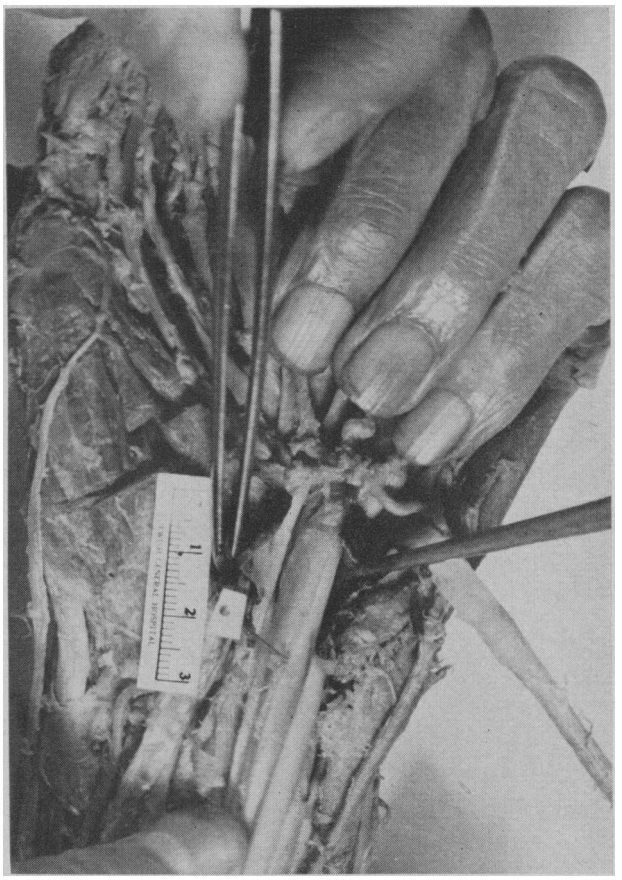

Fig. 2b (taken with fingers flexed). Showing that this fixed point has now moved proximal to the ligament (held with forceps).

tween the two operations. This was not because the tumour had grown so quickly—it was simply because there was now so much less tissue separating it from the surface, the transverse carpal ligament for the most part and some subcutaneous tissue having been cut through.

SUMMARY

A hitherto, to the author's knowledge, previously undescribed entity has been reported. Its relationship to the classical carpal tunnel syndrome has been described in some detail. Particulars of the case are given, and an illustration of the findings at operation is appended. The modus operandi of the tumour is explained from observations on the cadaver.

REFERENCES

1. ABBOTT, L. C. AND SAUNDERS, J. B. DEC. M.: *Surg. Gynec. & Obst.*, **57**: 507, 1933.
2. DE ABREU, L. B. AND MOREIRA, R. G.: *J. Bone & Joint Surg.*, **40-A**: 1426, 1958.
3. MERLE D'AUBIGNÉ, R. AND BÉNASSY, J.: *Mém. Acad. chir.*, **75**: 717, 1949.
4. BELL, G. E., JR. AND GOLDNER, J. L.: *South. M. J.*, **49**: 966, 1956.
5. BEYERS, A. J. AND KEET, M. P.: *South African M. J.*, **30**: 942, 1956.
6. BRAIN, W. R., WRIGHT, A. D. AND WILKINSON, M.: *Lancet*, **1**: 277, 1947.
7. BROOKS, D. M.: *J. Bone & Joint Surg.*, **34-B**: 391, 1952.
8. BROUWER, B.: *J. Nerv. & Ment. Dis.*, **51**: 113, 1920.
9. BUNNELL, S.: In discussion: Phalen, G. S.: *J. A. M. A.*, **145**: 1128, 1951.
10. CANNON, B. W. AND LOVE, J. G.: *Surgery*, **20**: 210, 1946.
11. FISHER, M.: *Canad. M. A. J.*, **71**: 121, 1954.
12. GILLIATT, R. W. AND WILSON, T.: *Lancet*, **2**: 595, 1953.
13. GROKOEST, A. W. AND DEMARTINI, F. E.: *J. A. M. A.*, **155**: 635, 1954.
14. HEATHFIELD, K. W. G.: *Lancet*, **2**: 663, 1957.
15. HIGHET, W. B.: *Lancet*, **1**: 227, 1943.
16. HUNT, J. R.: *Tr. Am. Neurol. A.*, **35**: 635, 1909.
17. *Idem*: *Rev. Neurol. & Psychiat.*, **12**: 137, 1914.
18. JENNY, F.: *Monatsschr. Psychiat. u. Neurol.*, **125**: 514, 1953.
19. KEITH, M. A.: *Scottish M. J.*, **2**: 435, 1957.
20. KELLGREN, J. H., BALL, J. AND TUTTON, G. K.: *Quart. J. Med.*, **21**: 405, 1952.
21. KENDALL, D.: *Brain*, **73**: 84, 1950.
22. KREMER, M. *et al.*: *Lancet*, **2**: 590, 1953.
23. KREMER, M. *et al.*: *Ibid.*, **2**: 590, 1953.
24. LIPSCOMB, P. R.: Tenosynovitis of the hand and the wrist: Carpal tunnel syndrome, de Quervain's disease, trigger digit, *In*: Clinical orthopedics. No. 13, edited by A. F. De Palma, J. B. Lippincott Company, Philadelphia and Montreal, 1959, p. 164.
25. LIST, C. F.: *Deutsche Ztschr. f. Nervenh.*, **124**: 279, 1932.
26. MACINTOSH, R. R. AND MUSHIN, W. W.: Local analgesia: brachial plexus, E. & S. Livingstone, Edinburgh and London, 3rd ed., 1954, p. 51.
27. MARIE, P. AND FOIX, C.: *Rev. neurol.*, **21**: 647, 1913.
28. MEADOFF, N.: *California Med.*, **70**: 252, 1949.
29. MOERSCH, F. P.: *Proc. Staff Meet. Mayo Clin.*, **13**: 220, 1938.
30. NEWMAN, P. H.: *Postgrad. M. J.*, **24**: 264, 1948.
31. NISSEN, K. I.: *J. Bone & Joint Surg.*, **34-B**: 514, 1952 (abstract).
32. PAGET, J.: Lectures on surgical pathology, Lindsay and Blakiston, Philadelphia, 1854, p. 42.
33. PHALEN, G. S.: The carpal tunnel syndrome, American Academy of Orthopedic Surgeons, Instructional Course Lectures, J. Edwards, Publisher, Ann Arbor, Mich., Vol. 14, 1957, p. 142.
34. PHALEN, G. S.: *J. A. M. A.*, **145**: 1128, 1951.
35. ROWNTREE, T. J.: *J. Bone & Joint Surg.*, **31-B**: 505, 1949.
36. SCHILLER, F. AND KOLB, F. O.: *Neurology*, **4**: 271, 1954.
37. SCHULTZE, F.: *Deutsche Ztschr. Nervenh.*, **3**: 300, 1893.
38. STEIN, A. H., JR.: *Missouri Med.*, **55**: 1197, 1958.
39. STEPHENS, J. AND WELCH, K.: *A.M.A. Arch. Surg.*, **73**: 849, 1956.
40. TINEL, J.: *Presse méd.*, **23**: 385, 1915.
41. VAINIO, K.: *Acta rheumat. scandinav.*, **4**: 22, 1958.
42. VICALE, C. T. AND SCARFF, J. E.: *Tr. Am. Neurol. A.*, **76**: 187, 1951.
43. WALLACE, J. AND COOK, A. W.: *Am. J. Obst. & Gynec.*, **73**: 1333, 1957.
44. WARD, L. E., BICKEL, W. H. AND CORBIN, K. B.: *J. A. M. A.*, **167**: 844, 1958.
45. WARTENBERG, R.: *Arch. Neurol. & Psychiat.*, **42**: 373, 1939.
46. WATSON-JONES, R.: *J. Bone & Joint Surg.*, **31-B**: 560, 1949.
47. *Idem*: Fractures and joint injuries, E. & S. Livingstone, Edinburgh, Vol. I, 3rd ed., 1943, p. 116.
48. WILSON, S. A. K.: *Proc. Roy. Soc. Med.*, **6**: 133, 1913.
49. WOLTMAN, H. W.: *Arch. Neurol. & Psychiat.*, **45**: 680, 1941.
50. WORMSER, P.: *Fortschr. Neurol. Psychiat.*, **18**: 211, 1950.
51. ZACHARY, R. B.: *Surg. Gynec. & Obst.*, **81**: 213, 1945.